Somatic Cell Counts in Bovine Milk: Relationships to Production and Clinical Episodes of Mastitis

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ABSTRACT

The relationships between somatic cell counts, milk production and episodes of clinical mastitis were evaluated using data collected between 1979 and 1981 in 32 southern Ontario Holstein herds. Somatic cell counts were logarithmically transformed and the distribution of the resulting counts is presented. The seasonal pattern in cell counts was evaluated using a formal statistical procedure. Counts were lowest in the winter and spring and highest in the early fall but the differences amongst monthly geometric mean cell counts were small.

Assuming a linear relationship between log somatic cell counts and test day milk production it was found that a unit increase in the log count resulted in a loss of 1.44 kg of milk. Regression analyses within specific log cell count ranges indicated that the previous estimate may underestimate losses at low cell counts and overestimate losses at higher cell counts.

The relationships between cell counts and episodes of mild or acute clinical mastitis were evaluated by comparing counts preceding and following the clinical episodes to comparable counts in matched control cows. Mild cases of mastitis were preceded by higher cell counts than were found in control cows but the same phenomenon was not observed in acute cases of mastitis. Both mild and acute cases were followed by higher cell counts than were found in control cows.

Key words: Mastitis, somatic cell count, production, relationship, season, distribution, case-control, dairy.

RÉSUMÉ

Cette étude consistait à évaluer les relations entre la numération des cellules somatiques, la production de lait et les épisodes de mammite clinique, à l'aide des données colligées, de 1979 à 1981, dans 32 troupeaux Holstein du sud de l'Ontario. Les auteurs transformèrent à cette fin les numérations des cellules somatiques en chiffres logarithmiques et ils présentent la distribution des valeurs qui en résultèrent. Ils évaluèrent aussi le profil saisonnier des numérations cellulaires, à l'aide d'un procédé statistique formel. C'est en hiver et au printemps que ces numérations s'avérèrent les plus basses, tandis qu'elles atteignirent un sommet, au début de l'automne; les différences entre les numérations cellulaires moyennes géométriques mensuelles se révélèrent cependant minimes.

En supposant une relation linéaire entre des numérations logarithmiques de cellules somatiques et la production lactée du jour de l'épreuve, on constata qu'une augmentation d'une unité dans la numération logarithmique résultait en une perte de 1,44 kg de lait. Des analyses de régression, à l'intérieur de l'éventail des numérations cellulaires logarithmiques spécifiques révélèrent que l'approximation précitée peut sous-estimer les pertes qui accompagnent les numérations cellulaires faibles et surestimer celles qui accompagnent les numérations cellulaires élevées.

L'évaluation des relations entre les numérations cellulaires et les épisodes de mammite clinique bénigne ou aiguë s'effectua en comparant les numérations antérieures et ultérieures aux épisodes de mammite clinique, avec des numérations équivalentes, chez des vaches témoins assorties. Les cas bénins de mammite furent précédés par des numérations cellulaires plus élevées que celles qu'on enregistra chez les vaches témoins; ce phénomène n'accompagna toutefois pas les cas de mammite aiguë. Les numérations cellulaires ultérieures aux cas bénins et aigus s'avérèrent plus élevées que celles des vaches témoins.

Mots clés: mammite, numération des cellules somatiques, production, relation, saison, distribution, contrôle de cas, laiterie.

INTRODUCTION

As the availability and use of individual cow somatic cell counting programs grows there is a need for a fuller understanding of how to interpret such counts and their relationship to level of milk production. Several possible transformations of somatic cell counts have been examined and it has been suggested that a logarithmic transformation ($\log_e$) is appropriate for improving interpretability (1). The possibility of adjusting cell counts for seasonal effects has also been considered but a careful evaluation of seasonal patterns has not been performed. This paper presents the distribution of cell counts following a logarithmic transformation and provides a statistical evaluation of seasonal patterns.

It is generally accepted that subclinical mastitis results in reduced milk production in dairy cows. In fact, although individual cow somatic cell

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counts have been used to differentiate between infected and noninfected cows (2) the relationship between the cell count and level of milk production may be of more importance to the dairyman. Knowledge of this relationship is essential in order to evaluate the significance of a subclinical mastitis problem in a herd or to carry out benefit/cost analyses of proposed control programs. This paper evaluates the relationship between logarithmically transformed somatic cell counts and test day milk production using all available data and also data within specified cell count ranges.

One of the concerns in implementing a control program designed to reduce losses due to subclinical mastitis is that it may increase the risk of cases of acute clinical mastitis (particularly due to coliforms) (3). It has been shown that elevated somatic cell counts do provide some protection against experimental infection with mastitis pathogens (4). However, it has also been reported that the point prevalence of clinical mastitis is higher in herds with higher levels of subclinical mastitis (5) and that there is a higher incidence of clinical mastitis in the daughters of sires of heifers with high cell counts than in daughters of sires of heifers with low cell counts (6). This study relates cell counts to episodes of mild and acute clinical mastitis by comparing both preceding and following counts from cows experiencing an episode of clinical mastitis to counts obtained from matched control cows.

MATERIALS AND METHODS

A study designed to evaluate relationships amongst diseases, production and survivorship was carried out in 32 southern Ontario Holstein herds between February 1979 and August 1981. Details of the project have been reported elsewhere (7,8). During the project, health, fertility and production data were recorded for 2876 lactations in 2009 cows.

In addition to data about clinical diseases, milk samples were collected for testing for subclinical ketosis and subclinical mastitis. For the eight herds closest to the Ontario Veterinary College (OVC), composite (cow) milk samples were collected on each visit by the production testing fieldmen (approximately 10-12 times per year). For the remaining 24 herds samples were collected four times per year. All samples were refrigerated and shipped fresh to the OVC. Upon arrival the samples were tested for ketone bodies using a qualitative nitroprusside based test (Ketotest, Denver Laboratories, Montreal, Quebec) with the results being scored as negative, + 1 or + 2. Samples were then fixed with formalin, incubated and the somatic cell concentration determined by a Coulter Milk Cell Counter (Coulter Electronics Inc., Hialeah, Florida). All somatic cell counts were transformed to a logarithmic scale (LSCC = $\log_e$ (somatic cell count x 10^{-3})).

Test results were merged with the health, fertility and production data by a series of interactive computer programs written in APL (a programming language) by the senior author. Analysis of the data was performed using the Statistical Package for the Social Sciences (9) and by a series of analytical programs written in APL.

DISTRIBUTION OF SOMATIC CELL COUNTS

The distribution of the cell counts in 1/2 $\log_e$ ranges was determined. Counts were then grouped according to the month in which the sample was collected and the geometric mean cell count for each month calculated. Within each month the proportion of cell counts exceeding 200,000 cells/mL was determined and the distribution of those proportions evaluated using Walter and Elwood's test for the seasonality of events (10).

RELATIONSHIP TO PRODUCTION

Least squares multiple linear regression was used to evaluate the relationship of the $\log_e$ somatic cell count (LSCC) to test day milk production according to the model:

$$Y_{ijk} = u + k_i + h_j + B_1(a_{ijk}) + B_2(d_{ijk}) + B_3(d_{ijk}^2) + B_4(p_{ijk}) + B_5(s_{ijk}) + \epsilon_{ijk}$$

Where Y_{ijk} = milk production in kg for the k^{th} observation in the j^{th} herd in the i^{th} ketone score
u = population mean
k_i = fixed effect of the i^{th} ketone score
h_j = fixed effect of j^{th} herd
B_1 to B_5 = regression coefficients for the age at calving (a_{ijk}), days in milk (d_{ijk} and d_{ijk}^2), previous lactation milk production (P_{ijk}) and the $\log_e$ somatic cell count (s_{ijk})
ϵ_{ijk} = random error term

Initially the model was evaluated using all of the observations for which there was complete data. Subsequently, the observations were divided into three subgroups according to the LSCC (LSCC < 5.0, $5.0 \leqslant$ LSCC < 6.0 and LSCC $\geqslant 6.0$) and the model evaluated within each category. Equality of the regression equations was tested using standard regression techniques (11).

CASE-CONTROL STUDY

Episodes of clinical mastitis occurring during the study were identified and classified as acute or mild depending on whether or not the cow required systemic therapy (e.g. antibiotics, fluids or steroids) in addition to local therapy administered in the udder. For each cow experiencing mild mastitis during a study lactation (i.e. a case cow) a control cow was selected from the same herd. The control cow was the cow closest in age to the case cow, which had not experienced any mastitis during the corresponding lactation. A cow could only serve as a control for one case. The number of days from calving to the first diagnosis of clinical mastitis in the case cow was calculated and all somatic cell counts determined between calving and the time of that diagnosis were recorded. Similarly, all cell counts determined in the control cow within the same time period postpartum were recorded and these counts are referred to as "previous" cell counts. In addition all cell counts determined in both the case and control cows between the time postpartum of the episode of clinical mastitis in the case cow, and the end of the lactation were recorded. These counts are referred to as "following" cell counts. For example: if clinical mastitis was first observed in the case cow at 87 days postpartum, then cell counts determined within the first 87 days of the lactation in both the case and control cow were recorded as "previous"

cell counts. Cell counts determined between day 88 and the end of the lactation in both cows were recorded as "following" cell counts.

Several comparisons between the $\log_e$ cell counts in case and control cows were made and their statistical significance evaluated by a paired Student's t test (12). The last "previous" cell counts (i.e. the LSCC determined closest to the time at which the clinical episode occurred) from case and control cows were compared. This comparison was carried out initially using all possible pairs of observations and subsequently using only those observations recorded within 60 or 30 days prior to the clinical episode. The averages of all "previous" counts were also compared. "Following" LSCC were compared in a similar manner using the next "following" LSCC (all observations or only those recorded within 60 or 30 days of the clinical episode). The averages of all "following" LSCC were compared.

The entire procedure was repeated for cases of acute mastitis and their controls. However, since acute episodes of mastitis generally occur early in a lactation there were insufficient counts available to obtain average "previous" values. Consequently, this comparison was not made.

RESULTS

DISTRIBUTION OF SOMATIC CELL COUNTS

A total of 14,590 milk samples were tested and the distribution of the $\log_e$ somatic cell counts is shown in Fig. 1. The range of 4.0 to 6.0 incorporated 72.3% of all the LSCC.

The geometric mean somatic cell counts and the proportion of counts over 200,000 cells/mL in each month are shown in Table I. The test for the seasonality of the proportions indicated that the centre of gravity of the counts was at 248.2° with January 1 representing 0°. This indicates that cell counts peaked in early September and the χ^2 test for the significance of this seasonal distribution was highly significant ($\chi^2 = 23.3$ with 2 d.f.; $p < 0.001$). However, the χ^2 test for departure from a unimodal pattern was also highly significant ($\chi^2 = 63.6$ with 11 d.f.; $p < 0.001$) indicating that the hypothesized unimodal seasonal distribution was not adequate to describe the data.

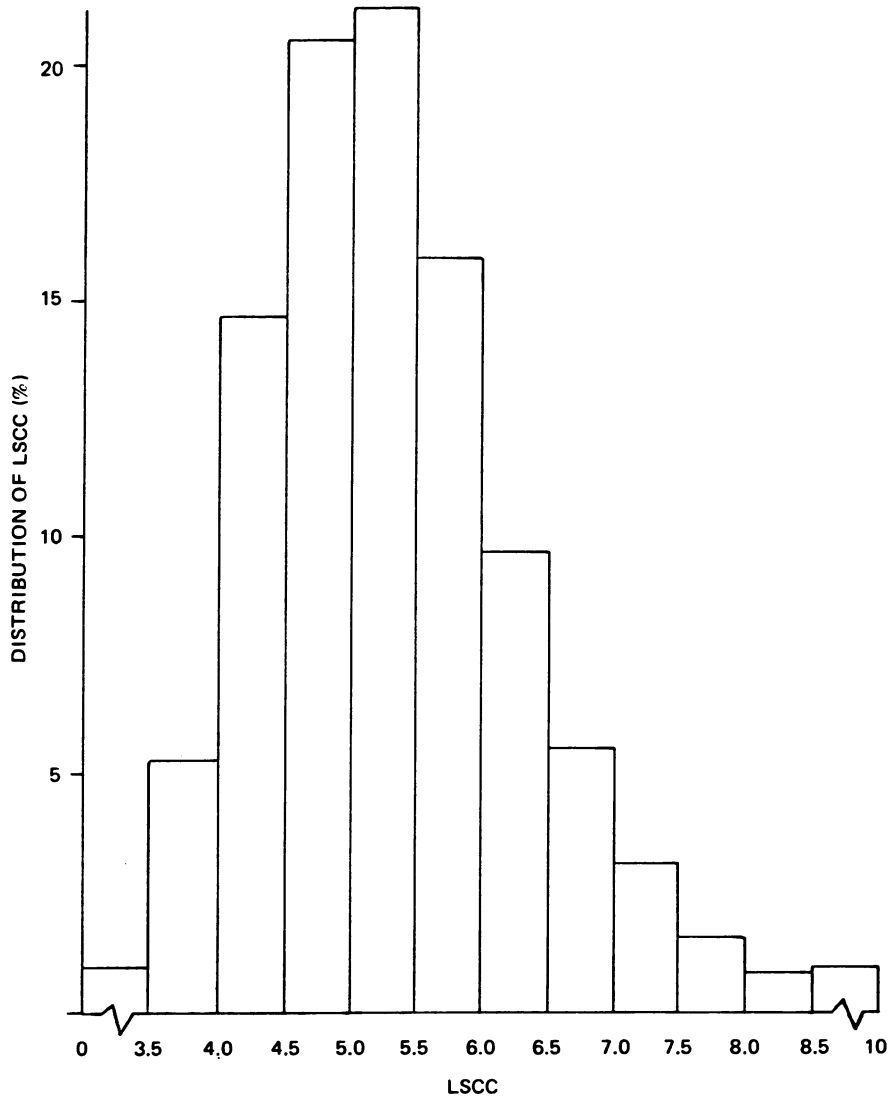

Fig. 1. Distribution of $\log_e$ somatic cell counts (LSCC) by 1/2 $\log_e$ ranges. Data from 14,590 somatic cell counts from 32 southern Ontario Holstein herds (1979-1981).

RELATIONSHIP TO PRODUCTION

Results of the evaluation of the relationship between LSCC and test day milk production are shown in Table II. As the regression function contained a term for milk production in the previous lactation, heifers were excluded from the analysis. Complete information was available for 6239 milk samples. The largest reduction in milk production was associated with LSCC

TABLE I. Seasonal Distribution of 14,590 Somatic Cell Counts Obtained from 32 Southern Ontario Holstein Herds (1979-1981)

Month	Geometric Mean Somatic Cell Count	Proportion of Counts over 200,000 (%)	Month	Geometric Mean Somatic Cell Count	Proportion of Counts over 200,000 (%)
January	199	42	July	219	48
February	175	38	August	222	47
March	201	44	September	245	53
April	222	51	October	229	52
May	178	39	November	199	44
June	215	47	December	205	45

TABLE II. Results from Regression Analyses of Test Day Milk Production on the Log_e Somatic Cell Count (LSCC). Data from 32 Southern Ontario Holstein Herds (1979-1981)

Somatic Cell Count Original Data (cells/mL)	LSCC	Number of Observations	B[a] for LSCC	Multiple R^2
< 149,000	< 5.0	2203	-1.80[b]	0.69
149-403,000	5.0-6.0	2519	-2.09[b]	0.70
⩾ 403,000	⩾ 6.0	1516	-1.21[b]	0.68
all	all	6239	-1.44[b]	0.71

[a]Unstandardized regression coefficient
[b]Sig. at $p < 0.0001$

between 5.0 and 6.0 with an intermediate loss associated with LSCC less than 5.0 and the smallest loss found when LSCC exceeded 6.0. The F statistic for testing the equality of the regression functions was highly significant ($F = 58.9$ with 4,6233 d.f.; $p < 0.01$) indicating that the regression lines in the three subgroups were not equivalent. Based on all observations, the estimated loss in milk production attributable to a unit increase in the LSCC was 1.44 kg or 6.1% of the mean daily milk production (23.5 kg).

CASE-CONTROL STUDY

The results of the case-control study for episodes of mild mastitis and acute mastitis are presented in Tables III and IV respectively. Cell counts were higher in cows about to experience an episode of mild clinical mastitis than in the matched control cows and the magnitude of the difference between the two groups increased as the period of observation was restricted closer to the time postpartum at which the episode occurred. In addition, cell counts remained higher following the clinical episode in those case cows than they did in the control cows and again the magnitude of the differences was greatest if the period of observation was restricted to 30 days following the time postpartum at which the clinical episode occurred.

In the analysis involving cows which experienced an episode of acute clinical mastitis, "previous" cell counts appeared to be lower in case cows than in control cows, but the difference was not statistically significant. However, the case cows did have significantly higher "following" counts than the control cows with the largest difference being observed when the period of observation was restricted to 30 days following the time postpartum at which the clinical episode occurred.

DISCUSSION

DISTRIBUTION OF COUNTS

It has been reported that a logarithmic transformation of somatic cell count data most nearly results in the data having the desired characteristics of normality and homoscedasticity among subgroups (13). In this study the log_e transformation was used and visual inspection of Fig. 1 suggests that the distribution of the LSCC was approximately normal. It is also evident that in this population of cows over 70% of the LSCC fall between 4.0 and 6.0 (approximately equivalent to 50,000 to 400,000 cells/mL), indicating the importance of understanding the relationship between cell counts and milk production in this range of counts.

The effects of season on somatic cell counts have been reviewed (14) and counts are generally reported to be highest in the summer and lowest in the winter. In this study the lowest

TABLE III. Geometric Mean Somatic Cell Counts Observed in Cows Experiencing (Cases) or Not Experiencing (Controls) an Episode of Mild Mastitis

Observation Period	Number of Observations	Geometric Mean Somatic Cell Count		Significance of Difference
		Case	Control	
Closest "previous" count				
— all observations	153	252	151	< 0.001
— only counts recorded within 60 days	68	310	156	< 0.001
— only counts recorded within 30 days	26	291	124	< 0.01
Average of "previous" counts	153	222	140	< 0.001
Closest "following" count				
— all observations	348	287	147	< 0.001
— only counts recorded within 60 days	170	358	147	< 0.001
— only counts recorded within 30 days	55	409	163	< 0.001
Average of "following" counts	348	277	173	< 0.001

TABLE IV. Geometric Mean Somatic Cell Counts Observed in Cows Experiencing (Cases) or Not Experiencing (Controls) an Episode of Acute Mastitis

Observation Period	Number of Observations	Geometric Mean Somatic Cell Count		Significance of Difference
		Case	Control	
Closest "previous" count				
— all observations	23	160	211	n.s.
— only counts recorded within 60 days	11	142	373	n.s.
— only counts recorded within 30 days	6	247	353	n.s.
Closest "following" count				
— all observations	62	401	192	< 0.001
— only counts recorded within 60 days	25	588	246	< 0.025
— only counts recorded within 30 days	8	592	156	< 0.01
Average of "following" counts	62	374	200	< 0.001

n.s. = not significant at $p = 0.05$

geometric mean cell counts were found in February and May and the highest in September and October. The Walter and Elwood's test for seasonality of events was used to evaluate the possibility of a unimodal (one maximum and one minimum per year) distribution of the proportion of cows having counts over 200,000 cells/mL. This procedure evaluates the magnitude of the seasonal effect as well as testing whether or not the distribution is unimodal and it can be applied to data with variable numbers of observations per month. The distribution of somatic cell counts was found to significantly deviate from a unimodal one, primarily due to the unexplained increase in the proportion of samples with elevated counts in April. However, the centre of gravity of the distribution (the point at which the largest proportion of elevated counts would be expected) was located in early September. Conversely, the lowest proportion would be expected in March.

It is not known if the seasonal pattern in cell counts is due to a physiological variation in cows, resulting in the excretion of more cells in the summer and fall, or if it is due to an increased prevalence of subclinical mastitis at that time of year. In addition, the difference between the lowest and highest geometric mean somatic cell counts was only 70,000 cells/mL. Consequently, the season of the year should not be considered a major determinant of somatic cell counts in southern Ontario.

RELATIONSHIP TO PRODUCTION

The relationship between milk production and untransformed somatic cell counts is not linear (15, 16). If the loss is linear on a logarithmic scale then the coefficients for the effect of the LSCC on milk production should be equal for various ranges of somatic cell counts. In this study there was a significant difference amongst the regression functions obtained from the three LSCC ranges analyzed. However, this difference may have been due to a variety of possible factors and no simple method of testing the difference amongst single coefficients from a series of multiple linear regressions exists. However, although the coefficients for the LSCC were not identical they did appear to be similar. It has been reported that a linear relationship between a lactational measure of somatic cell concentration and lactation total milk production is adequate since the addition of quadratic and cubic terms to the regression functions is generally not statistically significant (16). It appears that a logarithmic transformation is appropriate for somatic cell counts when evaluating their impact on milk production, but the results of this study suggest that such a relationship may slightly underestimate losses associated with low cell counts (< 400,000 cells/mL) and slightly overestimate losses at higher cell counts. However, a linear function based on LSCC will certainly better reflect the relationship between cell counts and production than a linear function using untransformed data.

The loss in milk production associated with an increase in the somatic cell counts from 50,000 to 400,000 cells/mL (3.89 kg) was approximately double the loss reported due to the same increase in a previous study (1.87 kg) (15, 17). Losses due to an increase from 400,000 to 1,100,000 cells/mL were very similar in the two studies (1.21 kg and approximately 1.06 kg for the present and previous studies respectively). Overall the loss associated with a unit increase in the LSCC (1.44 kg or 6.1% of the mean production) was higher than losses reported in the previous study (0.98 kg or 3.9%), (15,17) or in the study based on a lactational measure of somatic cell concentration (270 kg/lactation or approximately 3.9% of mean production) (16). The present study dealt only with the relationship between cell counts and milk production in multiparous cows. However, it has been reported elsewhere that losses associated with a similar increase in cell counts in first calf heifers is smaller (16).

At low cell counts the negative association between milk production and cell count may result in part from a dilution effect of yield on the cell count. Inclusion in the model of a term for yield in the previous lactation will eliminate some or all of this effect. It has been shown that inclusion of a term for previous production has relatively little effect on the coefficient for the $\log_e$ somatic cell count (16). However, the term was included in all models analyzed in this study in order to minimize any possible confounding attributable to a dilution effect. Consequently, it can be concluded that most of the effect of increasing somatic cell counts observed in this study was in fact a detrimental effect attributable to those cell counts.

CASE-CONTROL STUDY

The results in Table III indicate that cows experiencing cases of mild clinical mastitis had higher somatic cell counts prior to the diagnosis of the clinical case than the control cows. The difference in the "previous" cell count between the case and the controls became larger as the period of observation was progressively shortened to 30 days. These results are in agreement with those from a previous study (18) in which cows classified as having subclinical mastitis were much more likely to develop clinical mastitis in the following 90 days than cows classified as healthy. It appears that cows which already have subclinical mastitis are at greater risk of developing mild clinical mastitis than are uninfected cows.

It is also evident that cell concentrations tend to remain high following episodes of mild clinical mastitis. As the period of observation was extended the difference between the case and control cows diminished but the difference was highly significant even after all cell counts measured during the remainder of the lactation were averaged and compared. It is possible that the therapy provided to a large proportion of the clinical cases failed to completely eliminate the infection or the cows were rapidly reinfected. Alternatively, the elevated counts may have resulted from damage to the mammary gland that was slow to heal even in the absence of infection. Persistence has been shown to be a prominent feature of *Staphylococcus aureus* infections (18,19). These associations of clinical mastitis with both higher "previous" and "following" cell counts can not be attributed to differences in age, herds of origin or stage of lactation between the case and control cows because these factors were all controlled by the matching procedure.

It has been reported that a higher proportion of cows which experienced

a case of acute mastitis were previously classified as healthy (as opposed to subclinically infected) than were cows experiencing a case of mild mastitis (60.1 and 47.4% respectively) (18). In this study there was no statistically significant difference between "previous" cell counts in cows experiencing acute mastitis compared to control cows. Since most cases of acute mastitis occur early in the lactation the sample size available for this analysis was small and this could have contributed substantially to the lack of statistical significance for the apparent difference. However, it should be noted that most of the apparent difference was due to higher cell counts in the control cows used in this analysis compared to the control groups used in other analyses. This difference may be explained by the pairs of cows in this analysis being substantially different from pairs in other analyses (e.g. older or from farms with a high prevalence of subclinical mastitis) in which case the apparent difference between the case and control cows may be a real one. Alternatively it may simply be due to sampling variability. The fact that most of the difference between the cases and controls is evident in the analyses restricted to observation periods of 60 or 30 days, with sample sizes of eleven and six respectively, suggests that sampling variability is the most likely explanation. If that is the case, then there is no evidence to support the hypothesis that low cell counts increase the risk of a cow developing acute mastitis.

The persistency of infections following cases of clinical mastitis has been reported to increase with the severity of the case (18). This study confirms that elevated cell counts persist following cases of acute mastitis and those cell counts appeared higher than comparable counts following episodes of mild mastitis. These elevated counts may have resulted from failure of the therapy to eliminate the infection, rapid reinfection of the udder or persisting damage to the udder in the absence of infection. Given the major detrimental impact of subclinical mastitis on milk production and survivorship (20), these data suggest the need for cohort studies to evaluate the long term productivity and survival of cows experiencing episodes of acute mastitis.

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